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SHORT REPORTS

Severe Dettol (chloroxylenol and terpineol) poisoning

Phenol poisoning is recognised as being serious, but chloroxylenol, a common constituent of proprietary disinfectants is regarded to be of low[1 2] to moderate[3] toxicity, and the Dettol label describes it as non-poisonous. Although reference is made to a personal communication about a patient who died of an air embolus after attempting to induce an abortion by instilling a chloroxylenol disinfectant into her uterus, we failed to find any reports of serious poisoning after oral ingestion of chloroxylenol. We report here such a case.

Case report

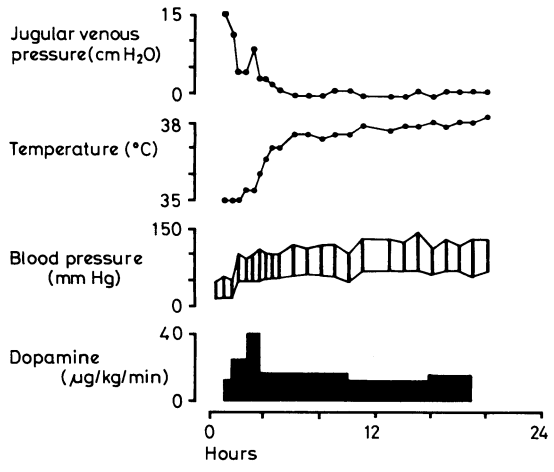
A 70-year-old depressed woman was admitted to the casualty department 30 minutes after attempting to commit suicide by ingesting 350 ml of household Dettol (Reckit and Colman), which contains chloroxylenol 48 g/l, terpineol, and ethyl alcohol (±7·0%).

She was in a deep coma, areflexic, and unresponsive to painful stimuli, and her pupils were moderately constricted showing little response to light. Her systolic blood pressure was 30 mm Hg, and her pulse 60 beats/min; she was breathing spontaneously at 8 per minute. Her rectal temperature was 35°C and she had signs of peripheral venous dilatation and a raised jugular venous pressure. There were no signs of corrosion or chemical irritation of the skin or mucosae and her ECG showed pronounced ischaemic changes.

An intravenous line was established, oxygen given via a face mask, and the stomach content carefully aspirated via a Ryles tube inserted through a nostril. The stomach content was milky and smelt strongly of Dettol. Careful gastric lavage was performed with small amounts of water, and afterwards 100 ml of liquid paraffin was left in the stomach. The patient was then transferred to the intensive care unit, where her vital functions were monitored continuously. Her blood pressure was controlled by dopamine infusion. About four hours after admission she developed a nodal tachycardia, which responded well to intravenous verapamil. After six to eight hours she started to regain consciousness and was fully conscious after 24 hours (see figure). She was then transferred to the general psychiatric ward and apart from severe watery diarrhoea during the first 48 hours recovered uneventfully.

Thin-layer chromatography of the gastric aspirate was compared with that of Dettol and its constituents and the presence of chloroxylenol and terpineol confirmed. Minute amounts of free chloroxylenol were present in the urine, but no chloroxylenol could be detected in the blood, although several phenolic compounds presumed to be metabolites and conjugation products were present. Large amounts of conjugated chloroxylenol were present in the urine.

Blood obtained on admission also showed an ethyl alcohol concentration of 700 mg/l and a sample of Dettol a concentration of 7·3%. Extensive gas-chromatography and thin-layer chromatography failed to show the presence of other potentially toxic substances.



Clinical course of patient during the first 24 hours.

Comment

This case has some interesting features. Apart from the rarity of the ingestion of such a large amount of this disinfectant (equivalent to 16·8 g of chloroxylenol), the rapidity of the development of profound central nervous system and cardiovascular depression and the remarkably rapid recovery are striking. The body seems to have very efficient mechanisms for rapidly detoxifying and eliminating chloroxylenol.

We thank the medical superintendent of the National Hospital, Bloemfontein, for permission to report this case.

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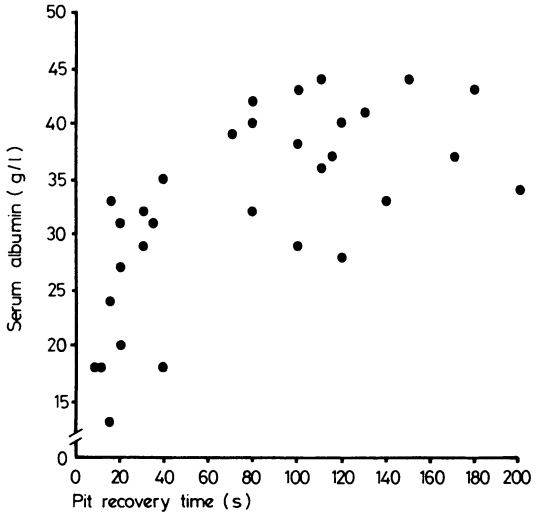
Assessment of hypoproteinaemic oedema: a simple physical sign

It is well known that hypoproteinaemia can cause pitting oedema. We have found that hypoproteinaemic oedema pits and recovers differently from other types of oedema but can find no reference to this in reports or texts on physical signs. We therefore investigated the relation between the rate of recovery of pitting and the serum albumin concentration in a group of oedematous patients.

Patients, methods, and results

Over four months all patients coming to our notice who had had pitting leg oedema for less than three months were studied. Their oedema was

assessed clinically, the recovery rate was measured, and venesection was performed without stasis for serum albumin estimation. All were seen before the diagnosis was ascertained. If the oedema over the tibia could be depressed deeply in one to two seconds by thumb pressure, and if a pit was observed to be visibly recovering two to three seconds after the release of pressure, the oedema was termed fast; if not it was termed slow. A device consisting of a perspex disc (45 mm diameter) with a Teflon-lined central ring containing a freely moving hollow perspex cylinder (15 mm diameter) was taped to the skin over the lower third of the tibia, with the patient semi-recumbent. The cylinder was depressed by firm digital pressure to a depth of 5 mm for 10 seconds, and the subsequent millimetre of recovery, measured visually on a Vernier scale, was timed after allowing two seconds for initial elastic recoil. The mean of two readings at different sites was taken. The consistency of the method was first established: no observations in a given patient differed from the mean by over 10%.



Relation between serum albumin concentration and pit recovery time in the 31 patients.

Thirty-one patients (15 men, 16 women) had enough oedema to be measured by the device; the results are shown in the figure. There was a significant relation between the pit recovery time and serum albumin concentration. By plotting log albumin concentration against log pit recovery time a regression coefficient was found ($P<0.001$). Clinical impressions of fast pitting oedema correlated with pit recovery times of 40 seconds or less.

Comment

Oedema has traditionally been divided into three main categories according to the mechanism of formation[1]: (*a*) congestive oedema, due to increased hydrostatic pressure, as in congestive cardiac failure and venous or lymphatic obstruction; (*b*) hypoalbuminaemic oedema, due to reduced plasma oncotic pressure, as in nephrotic syndrome, malnutrition, and malabsorption; and (*c*) capillary oedema, due to increased permeability of capillaries, as in vasculitis (including glomerulonephritis), and idiopathic oedema in women.

In hypoproteinaemic oedema the tissue fluid protein content is less than 1 g/l,[2] while in congestive cardiac failure it is 4-5 g/l.[3] In capillary oedema the protein content of the oedema fluid is higher, and is over 10 g/l[4] in glomerulonephritis. Moreover, the localised oedema produced by subcutaneously infused fluid can be dispersed more rapidly in patients with hypoalbuminaemia.[5]

The viscosity of tissue fluids is related to their protein content, and the mobility of pitting oedema presumably depends on this. The reduced protein content of the oedema fluid in hypoproteinaemia therefore explains the relation between the serum albumin concentration and the rate of recovery of pitting. This phenomenon could have a useful clinical application in that hypoproteinaemic oedema may be diagnosed by simple observation. When oedema pits with little resistance and recovery is visible in the initial seconds, hypoproteinaemia probably plays a part in the pathogenesis of the oedema and may be its major cause. In other types of oedema the pit tends to form less readily and recovers more slowly. Chronic oedema may not pit due to fibrosis and induration, but may pit rapidly due to laxity of the tissues.

We suggest that the nature of the pitting should be assessed in oedematous patients as it provides a physical sign which can be of value in making a clinical differential diagnosis.

We thank Mr A Johnson of the MRC Statistical Research Unit and Dr L J Grant for their help.

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Different periods of fasting have no effect on oral glucose tolerance

It has been suggested that the length of the pretest fast influences the result of an oral glucose tolerance test.[1] In that study the patients who exhibited least carbohydrate tolerance were woken at 0500 for a meal. It seemed possible, therefore, that factors other than the duration of the fast itself might have influenced the results obtained. To study the effect of fasting itself we altered the time of the test from 0900 to 1400. We also altered the longest period of fasting from 12 to 16 hours, thereby eliminating a disturbed sleep pattern as an integral part of the study.

Patients, methods, and results

A total of 23 consecutive adult patients admitted to the general medical wards without a past, present, or family history of diabetes who volunteered to participate in the study after its routine had been explained to them form the basis of this report. Each had three oral glucose tolerance tests performed after randomly selected fasting periods of four, eight, and 16 hours. The tests were all performed at 1400, on each of three days; each test was separated by not less than two days or more than five days. All were eating a normal hospital diet during the study. Fifty grams of glucose was given by mouth and venous blood samples taken at 0, 30, 60, 90, and 120 minutes. The blood glucose concentration was estimated on each specimen by Technicon method N-2b. One patient was discovered to have chemical diabetes and her results have been excluded from the table, which contains those for the other 22. The differences between the results were tested for significance by Student's *t* test.

The 30-minute values after the 4-hour fast were significantly higher than those obtained after either the eight-hour ($t=2.0195$, $0.05>P>0.02$) or the 16-hour ($t=2.0772$, $0.05>P>0.02$) fasts. No other differences were significant.

Mean (± SD of mean) blood sugar concentrations (mmol/l) for three oral glucose tolerance tests in 22 patients

Pretest fast (h)	Time (minutes)				
	0	30	60	90	120
4	4·30 ± 0·46	7·15 ± 1·24	7·14 ± 1·97	5·71 ± 1·43	4·81 ± 0·988
8	4·20 ± 0·31	6·45 ± 0·97	7·42 ± 1·62	6·33 ± 1·9	5·32 ± 1·56
16	4·28 ± 0·66	6·46 ± 0·88	6·52 ± 1·8	6·18 ± 2·04	5·42 ± 2·4

Conversion: SI to traditional units—Glucose: 1 mmol/l ≈ 18 mg/100 ml.

Discussion

While oral glucose tolerance may be less in normal people in the afternoon,[2] standardisation of the time of the three tests on each patient allows a valid comparison to be made between the results obtained in each of the tests. Unlike Walsh *et al*[1] we did not find that the duration of the pretest fast influenced the degree of carbohydrate tolerance. It is true that the values obtained at 30 minutes after the four-hour fast